Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of February 10, 1891.

The President, Dr. **Landon Carter Gray**, in the chair.

THE USE OF ATROPIA IN THE TREATMENT OF LOCALIZED MUSCULAR SPASM.

Dr. W. M. **Leszynsky** read a paper with this title. The speaker had in March, 1884, reported a case of clonic torticollis, successfully treated by the subcutaneous administration of the sulphate of atropine. The first case of the present series was that of a man, thirty-seven years of age, who had, three years previously, muscular spasm, beginning at the right side of the neck, which had come on six weeks after an abscess had been opened at the angle of the jaw on the right side. The force of the spasm was increased by mastication and closure of the jaw, and it was more active upon mental excitement. He was frequently aroused from sleep by the severity of the paroxysm. There was no complaint of pain, but only of numbness in the right lower lip. He was treated by means of medicine and electricity for four months without relief. There was well-pronounced clonico-tonic spasm affecting the right platysma, forcibly drawing downwards the facia about the chin and the lower angles of the mouth. The clonic character of the spasm preponderated, and was almost constant. The muscle was hypertrophied to nearly four times its normal size, and its outline was distinct and well defined.

There was an area of complete anæsthesia one and one-half by three centimetres at the vermilion border of the lower lip near the angle of the mouth on the right side, which extended inward over the mucous membrane and over the alveolar process of the lower jaw. The mechanical irritability of the facial muscles was well marked. The jaw jerk was demonstrable. There was no apparent caries of the teeth. There was no abnormality evident in other portions of the body. The treatment was commenced by the application of a cantharides plaster over the affected muscle. After one week had elapsed an injection of $^1/_{120}$ of a grain of sulphate of atropine was made into the platysma. For the next three days an injection of $^1/_{400}$ of a grain was given, when the exaggerated mechanical irritability of the facial muscles had markedly diminished and there was an abatement in the frequency of the spasm. Sensation was also returning in the anæsthetic area. The dose of atropine was gradually increased, until $^1/_{48}$ of a grain was given daily. At the end of two weeks the most energetic effort by pressure and otherwise had failed to produce any spasmodic action in the platysma. The administration of the atropine was then discontinued. Report from the patient a month later was that there had been no return of the spasm. The solution used was atropine sulph. gr. I to aq. distil. ounce I. One minim of this solution represented $^1/_{480}$ of a grain of atropine. The speaker's method had been to give four minims at the first injection and to be on the alert for constitutional effects. Upon discovering that there was no idiosyncrasy in regard to the drug, it was administered once daily, and the quantity gradually increased from day to day, until the desired effect was produced or intolerance was established. The production of severe constitutional symptoms was not an essential feature in this plan of treatment. One of the experimental physiological effects of the drug, when given in sufficient quantity, was to paralyze the motor nerves, first affecting their trunk.

Dr. V. P. GIBNEY, on being asked for his views on the subject of the paper, said he did not know whether the author had included in his earlier reports cases of hysterical torticollis, rotary spasm and so on. He doubted whether it was the intention to include that class of cases known as spastic paraplegias. The cases which came under his personal observation were only those of functional disturbance or deformity.

THE DEGENERATIVE DISEASES OF THE SPINAL CORD, WITH A DESCRIPTION OF A NEW TYPE

Title of paper read by Dr. Dana. See page 205.

Dr. B. SACHS said that at the present time it was difficult to agree or disagree with Dr. Dana. The clinical symptoms in his own and Dr. Putnam's cases had varied from the symptoms ordinarily met with in cases of combined sclerosis, but those who had seen many cases of tabes and of ataxic paraplegia and of other forms of combined scleroses would hesitate to create new types on the basis of a slight variation in symptoms. Moreover they now knew, from the investigations of many observers, that the degenerations which occurred in the spinal cord were not strictly symmetrical. These often overlapped the lines which divided the spinal tracts from one another. Cases had been recorded in which the degeneration of the white fibres had also involved the gray matter of the cord. He could not, therefore, see the advantages of creating a special type, because the degeneration in these cases happened to be somewhat differently distributed. He must object also to Dr. Dana's suggestion for the suppression of the term of compression myelitis. No doubt that many years ago the term was loosely applied and in a fair number of cases of compression of the cord the destruction of that organ was not the result of an inflammatory process. It was only within the last few months that Schmaus, in a series of beautiful researches, had demonstrated the extension of the inflammatory process from the bone to the spinal cord itself. In many cases of Pott's disease the symptoms might unhesitatingly be attributed to a true myelitic process.

Dr. G. R. ELLIOTT said that as to the possible pathology of the changes which took place in the spinal cord in the conditions under consideration, there had, he thought, been much useless discussion, as well as to the precise kinds of degeneration and inflammation which attacked nervous tissue. He thought that they might all agree that these changes were reconcilable with those which took place in other organs. In all processes recognized as inflammatory and running a protracted course, there was increase of connective tissue and results incidental to this. Nervous tissue had an intercellular substance the same as any other organ, with the same parenchyma and protoplasmic material, under certain modification. In the acute processes the cells underwent more decidedly rapid changes than was the case in the liver, kidneys, or heart. In the course of acute processes degeneration might or might not occur. Every process, whether degenerative or inflammatory, was, as he had said, followed by the increase of connective tissue, but the mere presence of this tissue in the nerve substance was no proof as to the initial lesion, whether inflammatory, degenerative or septic, or some process of softening, embolism or thrombosis. As to compression myelitis, there was no absolute proof to show that the lesion was inflammatory, and he thought everything pointed to the contrary. The examinations of the cords in these cases were usually made at a late stage, when the changes found and the increase of connective tissue present had given rise to the idea that the processes had been inflammatory from the beginning. Kohler had, however, demonstrated that similar changes could be brought about by mechanical pressure of the cord. In the majority of cases of compression myelitis following Pott's disease, there was no tendency for the inflammatory processes to attack any part of the cord. The inflammation was limited to a narrow site on the dura mater. It was only where the process was a rapid one and perforation of the dura ensued that leptomeningitis and myelitis was set up.

Dr. W. R. BIRDSALL thought that the position taken by Dr. Dana was correct, for he was not attempting to establish the existence of a new type of disease of the cord, but was merely calling attention to a group of symptoms differing in course from those of ordinary cases of combined sclerosis. In the opinion of the speaker, compression myelitis was not an inflammation of the cord in the majority of cases. He took exception to the view that placed changes in the cord on a par with those of other organs, and did not think they were justified in saying that the inflammatory processes in the cord and general nervous system were the same as those in other parenchymatous organs.

Dr. SACHS said that the remarks he had made in regard to compression myelitis appeared to have been misinterpreted. He was well aware that the term had often been misapplied, but the point he wished to make was, that in view of the very latest investigations, there was every reason to believe that a true myelitic process was responsible for the symptoms in a number of the compression cases, particularly in those in which a tuberculous process in the bone had been the starting point of the whole trouble.

Dr. HERTER had, in Pott's disease, seen direct extension of the inflammatory process from the dura mater to the cord, though in the majority of the cases examined post mortem, were seen at a time when changes in the cord were purely of a degenerative character and the inflammatory site was not to be seen.

The PRESIDENT said that it seemed to him that Dr. Dana had made a very useful contribution to the subject. Of course the paper was not tentative, drawing attention to what the author and others had observed. All this part of the spinal cord was a mysterious area, both clinically and pathologically. The author of the paper was perfectly warranted in throwing any possible light upon the subject, and in offering any suggestion based upon observation for the grouping of the pathological conditions and clinical symptoms in this part of the cord.

Dr. DANA thought that pathologists generally at the present time would agree with his views as to inflammation. Occasionally it arose from causes other than microscopic poisons, but there was usually some pyogenic germ as the starting point of these inflammations. If it was not possible to get any history pointing to the possible entrance of these, he believed it better to withhold any opinion as to the inflammatory or non-inflammatory nature of the lesion. Dr. Sach's suggestion that there might be tuberculosis of the cord at this level in some of these cases might be true, but in the great majority of the cases tuberculous meningitis would not end in sclerosis, but would probably destroy the cord by softening. He agreed that it was well to be cautious in making new types of disease ; still such conditions as Putnam had described were locomotor ataxias or ataxic paraplegias ; the prognosis and treatment were different. Prevention or early recognition were important. The conditions seemed to depend upon some toxic influence. In pernicious anæmia, the cord might take on degenerative changes, therefore great attention was necessary to the blood. Recognition of a type in disease was certainly of importance clinically.

Asylum Notes.

REPORT OF THE MICHIGAN ASYLUM FOR THE INSANE, 1890.

THE EVOLUTION OF AN ASYLUM.

BY WILLIAM D. GRANGER, M D.

This is the time of year for the appearance of asylum reports. That of the asylum at Kalamazoo is one of great interest and instruction.

The growth and changes in the institution since its opening in 1859, are clearly told by Dr. Palmer, the Superintendent, and the text is fittingly illustrated by ten excellent full page photographs.